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Effect of Intravenous Streptokinase on the Relation Between Initial ST-Predicted Size and Final QRS-Estimated Size of Acute Myocardial Infarcts

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Thrombolytic therapy has been documented to reduce acute myocardial infarct size. The previously established relation between initial ST segment elevation and final electrocardiographic (ECG) myocardial infarct size in patients without coronary reperfusion might therefore be altered by thrombolytic therapy. The effect of intravenous streptokinase on this relation was therefore studied in 73 patients with initial acute myocardial infarction who had participated in the Second International Study of Infarct Survival (ISIS–2). Patients who received streptokinase were considered as one group and patients who did not receive streptokinase as a control group. Final myocardial infarct size, which was estimated from the QRS score, was predicted from the admission standard ECG by previously developed formulas based on ST segment elevation.

In the 40 control patients there was no change from ST-predicted to final QRS-estimated infarct size (median 17.7% versus 18.3%; p = NS). In the 33 patients in the streptokinase group, there was a highly significant decrease from predicted to final myocardial infarct size (median 21.9% versus 16.2%; p < 0.0002). This decrease was found for both anterior (median 23.7% versus 19.5%; p < 0.03) and inferior (median 21.9% versus 12.0%; p = 0.001) infarct locations. Multiple regression analysis adjusting for differences in predicted infarct size confirmed the significance of streptokinase on the difference in infarct size (p = 0.006). Based on the variability of the percent change from predicted to final infarct size in the control group, a threshold decrease ≥20% is required for identification of salvage. Application of this threshold identified 10 (25%) of 40 control patients and 20 (61%) of 33 patients in the streptokinase group (p < 0.005).

Thus, intravenous streptokinase significantly changes the relation between predicted and final acute myocardial infarction size, demonstrating the potential of the standard ECG for noninvasive evaluation of myocardial salvage after thrombolytic therapy.

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Thrombolytic therapy has been shown to reduce final infarct size (1–3) in patients with acute myocardial infarction. In turn, reduction in infarct size has resulted in improvement of both left ventricular function and prognosis (4–7). Because thrombolytic therapy is therefore now routinely administered to patients with acute infarction, there is a need for noninvasive methods to evaluate its effectiveness.

Previous studies (8–10) have established the relation between the acute myocardial infarct size predicted by initial ST segment changes and that estimated by final QRS score in the absence of reperfusion therapy. The most quantitative of these studies (10) presented formulas describing this relation for both anterior and inferior acute infarct locations. In an independent study (11,12), the formula for anterior location was validated and that for inferior location modified to include consideration of the full spectrum of electrocardiographic (ECG) leads.

If the size of an acute myocardial infarction were limited by either spontaneous or therapeutic reperfusion, these

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Table 1. Electrocardiographic Criteria for Study Inclusion Based on Admission Epicardial Injury

≥1 mm ST↑ in one or more leads
Max ST↑ ≥ Max ST↓ unless the Max ST↓ is in leads V_1 to V_3
ST↑ in lead V_2 > ST↑ in lead V_4

ST↓ = ST segment depression; ST↑ = ST segment elevation; Max = maximal.

relations should be invalidated. The present study was performed to test this hypothesis in control versus streptokinase-treated groups of patients.

Methods

Study patients. A detailed description of patient enrollment in the Second International Study of Infarct Survival (ISIS–2) was recently published (5). The effect on mortality of intravenous streptokinase and oral aspirin treatment was studied in patients with suspected myocardial infarction who were admitted within 24 h of symptom onset (12 h in the Danish subpopulation). Patients were subsequently randomized to four treatment groups: 1) streptokinase alone, 2) aspirin alone, 3) both drugs, and 4) neither drug. The regimen for streptokinase (Streptase) was 1.5×10^6 U administered intravenously over 1 h and for aspirin 160 mg/day for 30 days. All patients admitted to Gentofte Hospital and Rigshospitalet with suspected myocardial infarction during 1986 and 1987 were considered for entry in the ISIS–2 trial in the absence of the following non-ECG exclusion criteria: contraindication to streptokinase or aspirin (history of stroke, gastrointestinal bleeding or ulcer, hypertension >200/100 mm Hg or recent severe trauma or operation) or the presence of other life-limiting disease.

Patients in the ISIS–2 trial were included in the present study if there was no ECG evidence of earlier myocardial infarction, bundle branch or fascicular block, ventricular hypertrophy or ventricular paced rhythm. Furthermore, ECG evidence of epicardial injury, as defined in Table 1, was required. Patients who received streptokinase alone or streptokinase and aspirin were considered as the streptokinase group; the patients who received aspirin or neither drug were considered as the control group based on the fact that the aspirin alone has not been considered capable of limiting myocardial infarct size.

Electrocardiographic considerations. The ECG showing the maximal ST segment deviation before treatment allocation was considered the initial recording and the final available in-hospital ECG was considered the discharge recording. The initial ECG was used to consider inclusion criteria (Table 1) and to classify the patients according to anterior or inferior infarct locations (Table 2). The study protocol stated that patients who had their maximal ST elevation in lead I, aVL or V_3 to V_6 would be grouped as having anterior or inferior myocardial infarction according to whether anterior or inferior sites had the greater ST elevation. Only one patient met this criterion.

Table 2. Acute Myocardial Infarct Location Criteria

Infarct Site	Criteria
Anterior	Max STΔ is ST↑ in leads V_1 to V_3
Inferior	Max STΔ is ST↑ in leads II, III, aVF, or ST↓ in leads V_1 to V_3

STΔ = ST segment deviation; other abbreviations as in Table 1.

The ST-predicted myocardial infarct (MI) size was determined from the initial ECG (Fig. 1 and 2). The following formulas based on the number of leads with ST elevation (↑) and the sum (Σ) of ST elevation were used for this prediction (10–12):

$$\text{Anterior \% MI size} = 3[1.5\ (\text{no. of leads with ST}\uparrow) - 0.4]$$

$$\text{Inferior \% MI size} = 3[0.6\ (\Sigma\ \text{ST}\uparrow \text{ in leads II, III, aVF}) + 2.0] + 3[1.5\ (\text{no. of noninferior leads with ST}\uparrow) - 0.4].$$

ST elevation was measured to the nearest 0.5 mm at the J point (10) in each lead except aVR. The formula for anterior myocardial infarction is used with confidence because it has been validated (11,12). This particular formula was selected for inferior myocardial infarction because it attained the highest correlation in both of the Danish populations in the study by Clemmensen et al. (12) However, it is used with caution because it was modified empirically and has not yet been validated in an extensive independent population.

The QRS-estimated myocardial infarct size was determined from the discharge ECG by using the Selvester QRS score (Fig. 1 and 2); this scoring system and its application mode have previously been described in detail (12,13). The system contains 54 criteria awarding a maximum of 32 points, each representing approximately 3% infarction of the left ventricle.

Data analysis. The initial ST segment–predicted infarct size and the final QRS-estimated size were determined for all patients with use of the methods previously described. These two expressions were compared for all patients and for the two infarct locations within each group. To provide a quantitative expression of the results of thrombolytic therapy, the difference between the initial ST segment–predicted myocardial infarct (MI) size and the final QRS-estimated size was determined for each patient as follows:

$$\text{Percent difference} = 100\ (\text{Final QRS-estimated MI size} - \text{Initial ST-predicted MI size}) \div \text{Initial ST-predicted MI size}.$$

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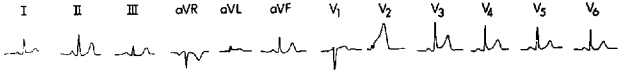
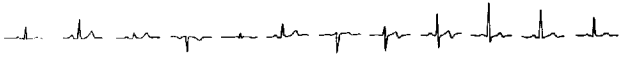


Initial ST-predicted MI size = 3[1.5(no. of leads with ST ↑) − 0.4]
= 3[1.5(6) − 0.4]
= 26%



Final QRS-estimated MI size = 3[QRS score]
= 3[1]
= 3%

$$\text{Percent difference} = 100 \times \frac{\text{Final QRS-estimated MI size} - \text{Initial ST-predicted MI size}}{\text{Initial ST-predicted MI size}}$$

$$= 100 \times \frac{3 - 26}{26}$$

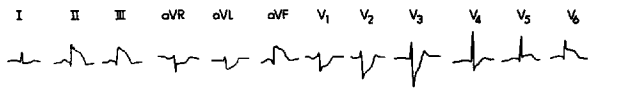
$$= -88\%$$

Figure 1. Twelve lead electrocardiograms from a patient with acute anterior myocardial infarction (MI). Calculation of initial ST-predicted infarct size, final QRS-estimated infarct size and percent difference is shown. ↑ = elevation.

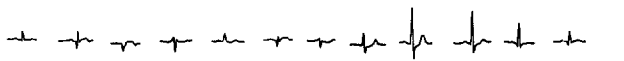
Use of this formula results in a negative percent difference in patients in whom the final QRS-estimated infarct size is smaller than the initial ST segment–predicted infarct size (Figs. 1 and 2) and a positive percent difference in patients whose final QRS-estimated infarct size is larger than the initial ST-predicted infarct size.

When the final QRS-estimated myocardial infarct size is smaller than the initial ST-predicted myocardial infarct size, it becomes important to identify the threshold beyond which myocardial salvage can be considered to have occurred. The formula for determining the percent difference in infarct size

Figure 2. Twelve lead electrocardiograms from a patient with acute inferior myocardial infarction (MI). Calculation of initial ST-predicted infarct size, final QRS-estimated infarct size and percent difference is shown. Σ = sum; other abbreviations as in Figure 1.



Initial ST-predicted MI size = 3[0.6(Σ ST ↑ in leads II, III, aVF) + 2.0] + 3[1.5(no. of noninferior leads with ST ↑) − 0.4]
= 3[0.6(21) + 2.0] + 3[1.5(2) − 0.4]
= 52%



Final QRS-estimated MI size = 3[QRS score]
= 3[9]
= 27%

$$\text{Percent difference} = 100 \times \frac{\text{Final QRS-estimated MI size} - \text{Initial ST-predicted MI size}}{\text{Initial ST-predicted MI size}}$$

$$= 100 \times \frac{27 - 52}{52}$$

$$= -48\%$$

Table 3. Estimated Difference Between Initial ST-Predicted Myocardial Infarct (MI) Size and Final QRS-Estimated Infarct Size

Group	N	Predicted MI Size (median %) (interquartile range)	Final MI Size (median %) (interquartile range)	% Difference*	p Value
Control	40	17.7 (14 to 25)	18.3 (12 to 29)	+ 3	NS
Anterior	17	16.9 (15 to 24)	20.4 (12 to 27)	+21	NS
Inferior	23	19.2 (14 to 25)	17.4 (12 to 36)	− 9	NS
Streptokinase	33	21.9 (17 to 33)	16.2 (10 to 22)	−26	<0.0002
Anterior	20	23.7 (17 to 30)	19.5 (13 to 22)	−18	<0.03
Inferior	13	21.9 (14 to 38)	12.0 (8 to 20)	−45	<0.001

*See Methods.

was first applied in the control group, and the value that identified the lowest quartile (below the 25th percentile) was identified and designated as this threshold. The proportions of patients in both control and streptokinase groups falling below this threshold of percent difference in myocardial infarct size were compared.

Statistics. Statistical analysis was performed using the Wilcoxon one-sample rank sum test and the Mann-Whitney two-sample rank sum test. Fisher's exact test examined differences in proportions. Multiple regression analysis that adjusted for differences in initial ST-predicted infarct size was used to assess the effect of infarct location and streptokinase treatment on the difference between initial ST-predicted size and final QRS estimated infarct size. Two-sided p values <0.05 were considered significant.

Results

The study group. Of the 120 patients randomized to the ISIS-2 trial at Rigshospitalet and Gentofte Hospital, 39 were excluded by ECG criteria and 8 were excluded by non-ECG criteria. The study final group consisted of 73 consecutive patients: 40 in the control group including 7 women (median age 58 years; range 36 to 71) and 33 in the streptokinase group including 5 women (median age 59 years; range 36 to 69). The median time to randomization was not significantly different between the control and streptokinase groups (2.8 h, range 1 to 12 versus 3.6 h, range 1 to 12). In the streptokinase group there was a trend toward earlier randomization in the inferior rather than in the anterior infarction group (3.3 h, range 1 to 7 versus 4.0 h, range 1 to 12; p = 0.09).

Initial predicted versus final estimated infarct size (Table 3). In the control group there was a nonsignificant (3%) increase from initial ST-predicted infarct size to final QRS-estimated infarct size. In the streptokinase group there was a highly significant (26%) decrease from the predicted to the final infarct size (p < 0.0002). The median final myocardial infarct size was slightly, but not significantly, smaller in the streptokinase group than in the control group (16.2% versus 18.3%, p = 0.22).

Anterior versus inferior infarction (Table 3). The patients receiving streptokinase in both infarct location groups had a significant decrease in final infarct size: inferior 45% (p = 0.001) and anterior 18% (p < 0.03). There were more patients with a larger initial ST-predicted infarct size in the streptokinase group. However, a multiple regression analysis that corrected for these differences confirmed the treatment effect of streptokinase (p = 0.006). This analysis also indicated that the infarct location had no significant effect on the difference between initial ST predicted and final QRS-estimated infarct size.

In the control group, patients in the lowest quartile had a ≥20% decrease from initial ST-predicted to final QRS-estimated infarct size (Fig. 3). Using this as the minimal threshold for detection, 20 (61%) of 33 patients in the streptokinase group versus 10 (25%) of 40 patients in the control group were considered to have had myocardial salvage (p < 0.005).

Discussion

Predicted and estimated infarct size. This study validates the previously established relation between initial ST-predicted myocardial infarct size and final QRS-estimated infarct size in control patients and demonstrates the beneficial effect in patients receiving thrombolytic therapy. The standard ECG used in this way may provide a new noninvasive approach for identifying patients who achieve myocardial salvage after thrombolytic therapy. One limitation is the necessity to exclude patients with factors such as bundle branch block and ventricular hypertrophy because the ECG indexes of ST-predicted and QRS-estimated myocardial infarct size have not been validated in their presence.

Comparison with previous studies on evaluating results of reperfusion therapy. The improved survival after thrombolysis for acute myocardial infarction is primarily attributed to reperfusion by way of a previously occluded coronary artery resulting in salvage of ischemic myocardium. The clinical

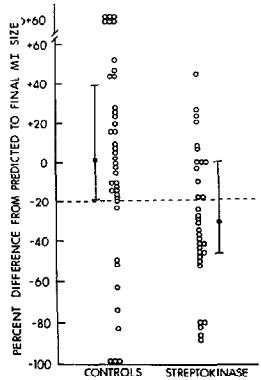


Figure 3. Each patient's (**open circles**) percent change from initial ST-predicted myocardial infarct (MI) size to final QRS-estimated infarct size in the control group (n = 40) and the streptokinase group (n = 33) is depicted. **Filled circles** represent the median and **bars** the 25th and 75th percentile levels for each group. The **broken line** at the 25th percentile level for the control group is the proposed threshold for detection of myocardial salvage. Salvage is attributed to patients with a difference from predicted to final infarct size on or below this line.

method for evaluation of the efficacy of therapy has been primarily cardiac catheterization, which is an invasive and costly technique. To overcome these limitations, several clinical studies have focused on the ability to noninvasively detect either myocardial reperfusion using various methods (14–19) or myocardial salvage using the standard ECG (8–11). The studies by Askenazi et al. (8) and Yusuf et al. (9) first demonstrated that the final myocardial infarct size could be predicted using the initial acute ECG in patients who did not receive size limiting therapy. They hypothesized that applying these methods in patients given such therapy could potentially assess the therapeutic effects. A more quantitative ST predictive method was recently developed by Aldrich et al. (10). This study, confirmed in an independent population (11), showed that the final QRS-estimated size of an initial myocardial infarction could be predicted from the ST segments on the admission ECG.

Use of the ECG to evaluate reperfusion therapy has also been proposed by Blanke et al. (20) using the somewhat different perspective of following the evolution of Q and R waves after reperfusion. Their study showed trends toward early (48 h postadmission) disappearance of Q waves and increase in R wave height in 15 patients with reperfusion by way of the left anterior descending artery. Their results confirm the observation of Albert et al. (21) comparing resolution of QRS changes after intraoperative myocardial infarction during coronary artery bypass grafting and after nonoperative myocardial infarction. In the operative group with the infarcted region reperfused by way of the graft, a significantly greater resolution of QRS changes, as quantified by the Selvester QRS scoring system, occurred from day 5 to 60 than in the nonoperative group. This difference persisted at 18 months' follow-up.

Myocardial salvage has also been suggested by investigators comparing QRS changes between groups of patients with or without infarct artery patency. Hackworthy et al. (22) found a significantly lower sum of Q waves in patients with reperfusion than in patients without reperfusion 24 h after treatment. Timmis et al. (23) found significantly more instances of non-Q wave myocardial infarction among patients with either spontaneous or pharmacologically induced reperfusion. In both of these studies (22,23) trends toward a smaller Selvester QRS score (24) were found in favor of patients with successful reperfusion. This is in accordance with the results of the present study of a trend toward a larger final QRS estimated myocardial infarct size in conventionally than in streptokinase-treated patients. Since myocardial salvage has been shown (2) to be enhanced only by early treatment with thrombolytic therapy, the relatively small difference in final QRS score in the two groups of this study could be due to inclusion of patients up to 12 h after symptom onset.

Anterior versus inferior infarction. The 26% overall reduction in infarct size in the streptokinase group agrees with a previous controlled study by Simoons et al. (2) that used the enzyme α-hydroxy-butyrate dehydrogenase (HBDH) for estimation of infarct size. A median 30% reduction in infarct size was observed in patients receiving intracoronary streptokinase compared with those treated conventionally. Bar et al. (25), also using HBDH, found approximately equal reduction of anterior and inferior myocardial infarct size (37% and 33%) in patients receiving either intracoronary or intravenous streptokinase when compared with control patients. In the present study there was an apparent difference in myocardial salvage in favor of inferior infarct location. That might result from earlier randomization in the inferior myocardial infarct group or from some inadequacy of the formula for estimating inferior infarct size. As stated in the Methods section, the formula for inferior myocardial infarct size used in the present study was empirically selected and has been validated only in a small test population; thus, it should be used with caution. It is reassuring that myocardial infarct location did not significantly affect the difference between predicted and final myocardial infarct size in the regression model.

Conclusions. This study suggests that myocardial salvage after thrombolytic therapy can be identified and possibly quantified noninvasively from the ECG in patients with an initial acute myocardial infarction, thus providing a simple technique to evaluate the therapeutic success of such therapy. This new diagnostic tool may be helpful in identifying patients with a large predicted myocardial infarct size who might benefit most from thrombolytic therapy and also in evaluating the clinical outcome of all patients who receive therapy aimed at myocardial reperfusion.

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